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Literature Review

Neuroanesthesia in Neurosurgical Emergencies: What Anesthesiologists Ought to Know

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Abstract

Neurosurgical emergencies demand rapid and coordinated anesthetic management to prevent secondary neurological injury and improve patient outcomes. Anesthesiologists play a critical role not only in facilitating surgical intervention but also in maintaining cerebral perfusion pressure (CPP), controlling intracranial pressure (ICP), and safeguarding neural function during periods of physiological instability. This review synthesizes current guidelines regarding anesthetic selection, hemodynamic targets, and specific neuroprotective strategies. Furthermore, optimal anesthetic management for acute ischemic stroke is re-evaluated based on recent research suggesting that conscious sedation may offer superior functional outcomes in minor anterior circulation strokes, whereas general anesthesia remains non-inferior for posterior circulation strokes. The review also highlights critical physiologic targets, emphasizing the strict avoidance of hypotension and the judicious use of moderate hypocapnia. By integrating these emerging evidence-based protocols, anesthesiologists can optimize management in neurosurgical emergencies settings, thereby improving patients' functional recovery and survival rates.

Keywords: Anesthesia, ischemic stroke, neuroprotection, neurosurgical procedures, TBIs (traumatic brain injuries)

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Introduction

Neurosurgical emergencies such as traumatic brain injuries (TBIs), brain herniation syndromes, ruptured intracranial aneurysms, acute spinal cord compression, and large vessel occlusion strokes represent some of the most time-sensitive and physiologically complex scenarios encountered in modern medicine. Without swift and coordinated intervention, these conditions can rapidly progress to brain herniation, irreversible neurological damage, or death.[1] In this critical period, anesthesiologists act not only as surgical support but also as integral members of the neurocritical team responsible for maintaining cerebral homeostasis, optimizing systemic physiology, and ensuring adequate neuroprotection from induction to emergence and beyond. The evolving landscape of neuroanesthesia emphasizes a shift toward evidence-based, protocol-driven approaches in the management of these emergencies. Growing literature underscores the importance of carefully controlling cerebral perfusion pressure (CPP) and intracranial pressure (ICP), as even minor deviations can lead to catastrophic outcomes in vulnerable neural tissue. Advanced intraoperative neurophysiologic monitoring (IONM)—such as somatosensory and motor evoked potentials (SSEP and MEP), bispectral index (BIS), and brain tissue oxygenation—has become increasingly incorporated to guide anesthetic depth and safeguard neural integrity.[2–5] Furthermore, anesthetic pharmacology continues to evolve with insights into the neuroprotective properties of agents such as propofol and dexmedetomidine. A 2023 meta-analysis[3],

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comparing dexmedetomidine and propofol during awake craniotomy showed dexmedetomidine provided comparable efficacy with potential advantages in surgeon satisfaction. However, this 2023 meta-analysis[3], which comprised only three clinical trials with a total of 138 patients, showed that this is still a new issue in neuroanesthesia. Furthermore, addressing other factors—including preoperative optimization, goal-directed fluid management, temperature control, and the judicious use of hyperosmolar therapies—is also recommended.[3,5] The anesthesiologist's role extends beyond intraoperative management to include perioperative decision-making, prognostic assessment, and contributions to neurocritical care pathways. In the face of life-threatening neurosurgical emergencies, a robust understanding of evidence-based neuroanesthesia is not optional, but essential.[4]

Importance of Emergency Neuroanesthesia

Neurosurgical emergencies represent a significant burden on healthcare systems worldwide and are associated with high morbidity, mortality, and long-term disability. In the pre-COVID-19 era, the Global Burden of Disease study estimated the global age-standardized incidence of TBI in 2016 at 369 cases per 100,000. Updated data for 2019 showed a slightly lower incidence of 346 cases per 100,000 population, with the highest incidence observed in low-and middle-income countries due to traffic accidents, falls, and violence.[6] Acute ischemic stroke, including those caused by large vessel occlusion remains the second leading cause of death globally, while subarachnoid hemorrhage from ruptured aneurysms contributes disproportionately to early mortality and long-term cognitive deficits. Spinal cord injuries and rapidly expanding intracranial masses such as epidural or subdural hematomas further compound the spectrum of emergencies requiring immediate neurosurgical and anesthetic intervention.[6] Anesthesiologists a play a crucial role in the acute management of these cases, not only facilitating surgery but also protecting the vulnerable brain and spinal cord during periods of profound physiological stress. The importance of neuroanesthesia in emergency settings lies in the delicate balance between supporting systemic circulation and preserving cerebral and spinal cord perfusion, while minimizing secondary insults such as hypoxia, hypercapnia, hypotension, and elevated ICP.[5] Emergency neuroanesthesia is a discipline that demands vigilant physiologic optimization, rapid decision-making, and a profound understanding of neurophysiology under duress. It bridges the gap between life-saving neurosurgical intervention and neurocritical care, with anesthesiologists playing a key role in influencing neurological outcomes from the first moment of airway control through the postoperative period. Understanding the pathophysiologic basis and evidence-based targets in cerebral perfusion, oxygenation, and pressure dynamics is essential for anesthesiologists tasked with these high-stakes cases.[4,5]

Anesthesia Consideration in Traumatic Brain Injury

The anesthetic management of patients with TBI revolves around two main goals: preventing secondary brain injury and facilitating urgent neurosurgical intervention. While the primary injury occurs at the time of trauma and is largely irreversible, secondary brain injury resulting from hypotension, hypoxia, hypercapnia, hyperthermia, and raised ICP is preventable and significantly influences neurological outcomes.[5]

Prevention of Secondary Brain Injury

Evidence shows that even a single episode of systolic blood pressure (SBP) <90 mmHg or a PaO_2<60 mmHg can double the mortality rate in patients with severe TBI.[2,7,8] Therefore, hemodynamic and respiratory stability must be prioritized from prehospital care through induction and into the intraoperative period. The Brain Trauma Foundation recommends maintaining SBP >100 mmHg in patients aged 50—69 and >110 mmHg in younger and older individuals.[2,5,7,8] Cerebral Blood Flow (CBF) is normally maintained through intrinsic autoregulatory mechanisms that adjust cerebrovascular resistance in response to changes in mean arterial pressure (MAP). However, in patients with TBI, ischemic

stroke, or increased ICP, this autoregulation can become impaired or even completely lost. The cerebral vasculature becomes pressure-passive under such conditions, meaning that CBF becomes directly dependent on systemic blood pressure. This loss of autoregulation increases the risk of both hypoperfusion and hyperemia, leading to ischemia, edema, or hemorrhagic transformation. To assess cerebral autoregulation, the anesthesiologist may increase MAP by 10–15 mmHg for less than 20 minutes. If ICP rises, it indicates impaired autoregulation (Figure 1).[4,5,9] Given these vulnerabilities, the anesthesiologist must prioritize the maintenance of CPP, calculated as the difference between MAP and ICP. The 2016 Brain Trauma Foundation guidelines recommend a CPP target of 60—70 mmHg in patients with severe TBI, as values below this range are associated with ischemic injury, while higher pressures may exacerbate cerebral edema or hemorrhage. Maintaining this delicate equilibrium requires close monitoring and frequent titration

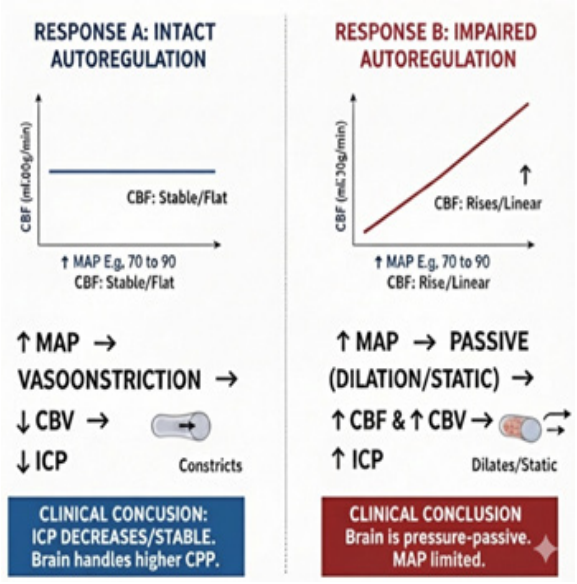

Figure 1. Hemodynamic response patterns during a Mean Arterial Pressure (MAP) challenge. (A) Intact Autoregulation: An elevation in MAP results in stable Cerebral Blood Flow (CBF) and stable or decreased Intracranial Pressure (ICP) due to reflexive vasoconstriction. (B) Impaired Autoregulation: The cerebrovascular bed becomes pressure-passive; an elevation in MAP leads to a linear increase in CBF and cerebral blood volume, resulting in a parallel rise in ICP

of fluids, vasopressors, and anesthetic depth.[2,8]

Choice of Induction Agents

The selection of induction agents is critical in these patients. Propofol is widely used for both induction and maintenance due to its favorable neuroprotective profile, as it reduces the cerebral metabolic rate of oxygen ($CMRO_2$), decreases ICP, and facilitates burst suppression in high doses. However, it may cause dose-dependent hypotension, particularly in patients with hypovolemia or impaired autoregulation. For hemodynamically unstable patients, Etomidate (0.2—0.3 mg/kg) remains a viable induction agent due to its cardiovascular stability and ability to reduce $CMRO_2$ and ICP. Though concerns about adrenal suppression exist, a single bolus has not been shown to significantly worsen outcomes in TBI.[4] Ketamine–once widely avoided in patients with TBI due to concerns that it might increase ICP–has undergone significant reevaluation in recent years.

Early studies suggesting ketamine-induced elevations in ICP were largely conducted in non-ventilated patients with poor baseline cerebral compliance. More recent evidence, however, indicates that in mechanically ventilated TBI patients with controlled ventilation and adequate sedation, Ketamine does not increase ICP and may even contribute to improved CPP through its sympathomimetic properties. These effects support systemic blood pressure and maintain cerebral autoregulation, which is often impaired in the setting of brain injury. Additionally, Ketamine's ability to prevent hypotension, a major contributor to secondary brain injury makes it particularly valuable in hemodynamically unstable patients where agents like propofol may induce profound vasodilation and hypotension. Furthermore, Ketamine exhibits NMDA receptor antagonism, which may confer neuroprotective effects by reducing excitotoxicity, a key mechanism of secondary neuronal injury in TBI.[2] This pharmacologic profile supports ketamine's use not only for induction but also for maintenance of anesthesia in TBI cases, especially when preserving MAP and CPP is critical. This

paradigm shift underscores the growing emphasis on individualized anesthetic management based on patient- specific physiology, rather than rigid adherence to outdated contraindications.[10]

Maintenance and Monitoring

During maintenance, a balanced anesthetic approach is preferred. Propofol infusions or low concentrations of volatile agents may be used, though volatile anesthetics can increase CBF and ICP at higher doses. Total intravenous anesthesia (TIVA) with propofol and short-acting opioids provides better control over hemodynamics and ICP. Neuromuscular blockade is often necessary to facilitate controlled ventilation and reduce metabolic demand. Continuous monitoring of end-tidal CO_2, arterial blood pressure, core temperature, and, when available, ICP, brain tissue oxygenation ($PbtO_2$), and jugular venous oxygen saturation ($SjvO_2$) are essential for guided neuroprotection.[3,5,6]

ICP Management and Hyperosmolar Therapy Acute management of increased ICP is one of the basic principles of anesthetic care in TBI. Hyperosmolar therapy using Mannitol at 0.25—1 g/kg IV bolus is traditionally employed to reduce ICP through osmotic diuresis. However, hypertonic saline, typically 3% solution at 2—5 mL/kg or via continuous infusion, has gained favor due to its superior ability to expand intravascular volume, sustain ICP reduction, and reduce the risk of rebound intracranial hypertension. Hypertonic saline also supports systemic hemodynamics and may be particularly beneficial in patients with coexisting hypotension. Serum sodium levels and osmolality must be closely monitored during administration to avoid complications such as osmotic demyelination syndrome.[5,11]

Other Strategic Measures

Several perioperative factors significantly influence neurological outcomes in patients with TBIs and must be carefully optimized. Ventilatory management is equally crucial. Hypoxia and hypercapnia are potent cerebral vasodilators and must be rigorously avoided, as they contribute to increased cerebral blood volume and ICP. Normoxia and normocapnia ($PaCO_2$ 35—45 mmHg) should be maintained through controlled ventilation, particularly in patients with herniation risk or diffuse cerebral edema. Hyperventilation, though sometimes necessary for acute ICP control, should be used with caution and only as a temporizing measure, as excessive hypocapnia may lead to cerebral ischemia. Moderate hypocapnia ($PaCO_2$ 30–35 mmHg) is recommended to minimize ischemic risk. Aggressive hyperventilation ($PaCO_2$ <30 mmHg) should be reserved as a rescue therapy if first-tier interventions (e.g., ventricular drainage and hyperosmotic agents such as Mannitol) fail.[2,5,7,8] Oxygenation should be closely monitored, with a target of normoxia or mild hyperoxia (PaO_2 >80 mmHg). Hypoxemia is a well-established predictor of poor neurologic outcome and must be aggressively prevented.[11,12]

Temperature control (36—37°C) is another critical aspect, as hyperthermia exacerbates neuronal injury through increased metabolic demand and excitotoxicity. Normothermia should be maintained using active cooling methods when necessary. Current evidence suggests that even mild fever in the early post-injury period is associated with worsened neurologic outcomes and higher mortality rates.[5,9] Glycemic control must also be addressed, as both hypoglycemia and hyperglycemia have deleterious effects on the injured brain. Studies recommend maintaining blood glucose levels within a moderate range of 110–180 mg/dL to reduce the risk of ischemic damage and metabolic stress, without increasing the risk of hypoglycemic episodes.[8,11]

Finally, seizure prophylaxis should be considered in patients at high risk, particularly those with cortical contusions, depressed skull fractures, or early post-traumatic seizures. Antiepileptic agents such as phenytoin or levetiracetam are commonly used. Current evidence suggests levetiracetam may be preferred due to a more favorable side effect profile and reduced risk of drug interactions.[8,11] Taken together, these measures form the foundation of a neuroprotective

strategy in the anesthetic management of TBI, emphasizing the importance of maintaining physiologic stability to mitigate secondary brain injury.

Anesthesia Consideration in Vascular Neurosurgery

The anesthetic management of patients undergoing emergent or urgent vascular neurosurgery, particularly for ischemic stroke and aneurysmal subarachnoid hemorrhage (aSAH), requires an approach focused on minimizing secondary injury, facilitating timely reperfusion or aneurysm control, and preserving neurophysiologic stability. Ischemic Stroke and Endovascular Therapy In acute ischemic stroke, every minute of delayed treatment increases the loss of viable brain tissue. Rapid revascularization, whether through intravenous thrombolysis with tissue plasminogen activator (tPA) or mechanical thrombectomy, is paramount in reducing infarct volume and improving outcomes. Per current AHA/ASA guidelines, patients eligible for intravenous thrombolysis must meet strict blood pressure criteria, with systolic BP<185 mmHg and diastolic BP <110 mmHg before tPA administration, and <180/105 mmHg for the subsequent 24 hours. These targets are based on the increased risk of hemorrhagic transformation associated with uncontrolled hypertension.[13]

For patients undergoing intra-arterial thrombectomy, the choice of anesthetic technique remains debated. Recent major randomized controlled trials—specifically GOLIATH, SIESTA, and ANSTROKE—demonstrated that general anesthesia is non-inferior and potentially superior in selected settings to monitored anesthesia care (MAC) or conscious sedation in terms of neurologic outcomes and procedural success. General anesthesia offers immobility, controlled ventilation, and improved procedural conditions, particularly in patients with agitation, poor respiratory effort, or posterior circulation strokes. However, when airway reflexes are intact and patient cooperation is achievable, MAC is often preferred to reduce induction time and hemodynamic fluctuations.[13-16] The optimal anesthetic strategy may depend on stroke severity and location. For anterior circulation stroke with mild symptoms (low NIHSS), a recent study found that conscious sedation (CS/LA) offered a more favorable risk-benefit ratio, showing significantly better 90-day functional outcomes than GA, despite GA achieving higher rates of complete recanalization.

However, in posterior circulation stroke, the superiority of CS/LA is less clear. An updated meta-analysis revealed that when baseline characteristics are adjusted, there are no significant differences between GA and CS/LA concerning functional independence, mortality, or successful reperfusion.[17,18] Recent evidence emphasizes the need for continuous blood pressure control, particularly during and after revascularization. Hypotension during thrombectomy is independently associated with worse functional outcomes, making intra-procedural BP targets, often >140 mmHg systolic, critical to maintain cerebral perfusion, especially in the penumbra. Sedation depth should be carefully titrated to avoid over-sedation that may precipitate hypoventilation or hypotension.[13]

Aneurysmal Subarachnoid Hemorrhage (aSAH) Management of aneurysmal SAH involves early rupture control, prevention of rebleeding, and mitigation of secondary complications such as cerebral vasospasm, hydrocephalus, and delayed cerebral ischemia. Preoperatively, patients require blood pressure management, typically maintaining systolic BP<160 mmHg to reduce the risk of aneurysmal re-rupture, while ensuring adequate cerebral perfusion. Nimodipine, a dihydropyridine calcium channel blocker, remains the only pharmacologic agent with proven efficacy in reducing poor outcomes from delayed ischemia and should be initiated in all patients unless contraindicated.[19] During aneurysm clipping surgery, meticulous anesthetic management is essential. A smooth induction with agents that minimize hemodynamic swings is critical to prevent surges in blood pressure, which could precipitate rebleeding. Volatile anesthetics, Propofol, or a balanced anesthetic technique can be used depending on

the surgical and physiologic context. Mannitol or hypertonic saline may be administered to reduce brain bulk and improve surgical exposure, particularly in cases of elevated ICP or significant mass effect.[20] Postoperatively, vigilant neurologic monitoring and blood pressure control continue to be paramount. In patients who undergo coiling or clipping, early detection and management of vasospasm, hydrocephalus, and electrolyte disturbances are critical aspects of care. Transcranial Doppler (TCD) ultrasonography may aid in vasospasm detection, while maintaining euvolemia and instituting hemodynamic augmentation may be necessary in cases of symptomatic delayed cerebral ischemia.[20]

Conclusions

The anesthesiologist's role in neurosurgical emergencies extends beyond facilitating surgery to actively preserving neurological function by preventing secondary brain injury. Principal management consists of strict physiologic targets, ranging from maintaining MAP to ensure adequate CPP and controlling ICP, to preventing hypoxia and hypercapnia. Extended strategies include the tailored choice of anesthetic agents in acute ischemic stroke to improve functional outcomes, as well as the early detection of complications following aneurysm clipping surgery. Moreover, ongoing research continues to explore novel strategies to optimize outcomes in neurosurgical emergencies.

AI Use Disclosure

Images within this manuscript were generated with the assistance of Gemini, a generative AI tool. The authors verify that all AI-generated content has been reviewed for accuracy and accept full responsibility for the integrity and originality of the published work.

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